LETTERS TO THE EDITOR

Scope

Heart welcomes letters commenting on papers published in the journal in the previous six months. Topics not related to papers published earlier in the journal may be introduced as a letter: letters reporting original data may be sent for peer review.

Presentation

Letters should be:

- not more than 600 words and six references in length
- typed in double spacing (fax copies and paper copy only)
- signed by all authors

They may contain short tables or a small figure. **Please send a copy of your letter on disk.** Full instructions to authors appear in the July 1999 issue of *Heart* (page 116).

Commotio cordis: sudden death due to chest wall impact in sports

EDITOR,—In his editorial,[1] Link comments that 70 deaths have been reported from commotio cordis while playing American sports since the late 1970s (mostly baseball, ice hockey, and American football), but there have been no reported deaths from this cause while playing cricket. Haq[2] raises the possibility of such a fatality occurring during a cricket match in Kentucky in 1970, but there are no reports of deaths occurring in Britain, where cricket is a popular summer sport. This may surprise many, as a cricket ball is of similar size, weight, and hardness to an American baseball, and commonly travels at similar speeds.

As Link suggests, there is probably a relative lack of awareness of the phenomenon of commotio cordis outside the United States. However, the dynamics of cricket are such that impacts of balls to the central chest of batsmen are rare. A baseball is "pitched" from a distance of 20 yards and aimed, without bouncing, to closely pass the batter between chest and knee height. In contrast, a cricket ball is "bowled" by a straight arm, windmill-type action from a distance of 22 yards and bounced once towards three 28" high wooden stumps, usually passing the batsman below waist level. This lower trajectory, as well as the more side on stance of the batsman, makes central chest impact by the ball considerably less frequent than in baseball. Only infrequently is the cricket ball bowled at chest height, either without bouncing (a "full toss", recently outlawed), or deliberately bounced fast and short to intimidate the batsman (a "bouncer"). The batsman's usual response is to take simple evasive action by ducking or swaying. The alternative is to play an aggressive shot in which the bat and shoulders (of a right handed batsman) are swung from right to left in a high horizontal arc, aiming to hook the ball from in front of the chest in an action broadly resembling a baseball shot. Only in this infrequent situation is the batsman likely to be struck in the central chest and placed at risk of commotio cordis. It is perhaps for this reason that chest protection is rarely worn by cricket batsmen, except when facing the fastest bowlers at the highest professional level.

Recent research has provided a better understanding of the dynamics of chest impact.[3] Increasing awareness of the dangers of low energy chest wall impact has encouraged use of appropriate protective measures and this will reduce the incidence of these most tragic sporting deaths.[4] Although there is less risk of commotio cordis in cricket than in baseball or ice hockey, the lessons learnt from American sports fatalities and the precautions necessary to safeguard the young participants are transferrable to other sports.

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1 Link MS. Commotio cordis: sudden death due to chest wall impact in sports. *Heart* 1999;**81**:109–10.
2 Haq CL. Sudden death due to low-energy chest-wall impact [letter]. *N Engl J Med* 1998; **339**:1399.
3 Link MS, Wang PJ, Pandian NG, *et al*. An experimental model of sudden death due to low-energy chest-wall impact (commotio cordis). *N Engl J Med* 1998;**338**:1805–11.
4 Abrunzo TJ. Commotio cordis: the single most common cause of traumatic death in youth baseball. *Am J Dis Child* 1991;**145**:1279–82.

Commotio cordis: a precordial thump?

EDITOR,—There has been growing interest in commotio cordis, defined as a rare type of sudden cardiac death after minor chest wall blows, mainly occurring in apparently healthy young people during sporting activity.[1–3] However, this event may be more common, but usually misunderstood and underreported, because of misclassification with other cardiac diseases in different settings and at older ages, in patients with associated symptoms.

About 10 years ago I admitted a man in his 50s without known cardiac disease to a coronary care unit for ECG monitoring. He reported a history of prolonged episodes of haemodynamically well tolerated palpitations, which had never been clearly diagnosed as symptoms always spontaneously disappeared before any ECG recording. During ECG monitoring he had sustained ventricular tachycardia (180 beats/min) with his usual palpitations. Before using any antiarrhythmic drugs, I tried to stop the ventricular tachycardia with a chest thump, as I had successfully done many times in similar situations. Surprisingly, my thump instantaneously transformed the well tolerated ventricular tachycardia into ventricular fibrillation with immediate loss of consciousness. Cardioversion (dc shock 320 J) immediately restored sinus rhythm and the patient resuscitated. I now consider any chest thump as a potentially dangerous and proarrhythmic manoeuvre.

It is quite surprising that this kind of commotio cordis (chest thump) is commonly adopted as an emergency therapeutic tool in patients with ventricular arrhythmias to reset myocardial electrical potentials, but the same phenomenon is considered a curious clinical finding when it accidentally happens during the vulnerable period of the cardiac cycle in healthy but unlucky young people (chest wall impact). Have we long been discussing the existence of an electromechanical phenomenon (commotio cordis) that we commonly exploit in emergency departments with another name and in different clinical setting (precordial thump)?

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1 Link MS. Commotio cordis: sudden death due to chest wall impact in sports. *Heart* 1999;**81**:109–10.
2 Link MS, Wang PJ, Pandian NG, *et al*. An experimental model of sudden death due to low energy chest wall impact (commotio cordis). *N Engl J Med* 1998;**338**:1805–11.
3 Maron BJ, Poliac LC, Kaplan JA, *et al*. Blunt impact to the chest leading to sudden death from cardiac arrest during sports activities. *N Engl J Med* 1995;**333**:337–42.

Contrast echocardiography during pericardiocentesis

EDITOR,—I read with interest the short case report by Betts and Radvan on the use of contrast echocardiography during pericardiocentesis.[1] I would like to suggest a much simpler and cheaper method of differentiating pericardial space from a cardiac chamber during pericardiocentesis.

For the past 45 years I have always included an ampul of dehydrocholate and an ampul of lobeline on the sterile pericardiocentesis tray.[2] Whenever the needle encounters blood or bloody fluid, one is faced with the problem of whether it is the pericardial cavity or a cardiac chamber that the needle tip has entered. Before aspiration is attempted, either dehydrocholate or lobeline in case of an obtunded patient should be injected. If the patient gives a typical response as in an ordinary circulation time determination, the needle tip must be in the cardiac chamber and should be withdrawn promptly. A negative response ensures an extracardiac location of the needle tip, and further aspiration or air injection for pneumopericardium (for assessment of the pericardial thickness) could be carried out with impunity. In parts of the world where dehydrocholate or lobeline might not be readily available, magnesium sulfate might be substituted.

A circulation time determination during pericardiocentesis provides a simple, safe, and accurate bedside method of differentiating between bloody pericardial fluid and intracardiac blood. It is also far less expensive than echocardiography. Furthermore, it is a much more expedient procedure during an emergency in suspected cardiac tamponade, especially when an echocardiographic machine is not readily available.

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1 Betts TR, Radvan JR. Contrast echocardiography during pericardiocentesis. *Heart* 1999;**81**:329.
2 Cheng TO. Ventricle or pericardial space? *Ann Intern Med* 1973;**78**:461.

This letter was shown to the authors, who reply as follows:

We note with great interest the suggestion of dehydrocholate injection as a way of differentiating the pericardial space from penetration into a cardiac chamber. It is an astute use of an old method of investigating for heart failure. Dehydrocholate, a bile salt, would be injected into a peripheral vein and the time taken for it to reach the tongue, when a bitter taste suddenly appeared, was measured as the circulation time, an index of ventricular function. Although we applaud its ingenuity we cannot recommend it as a substitute for more modern techniques. It is the advent of echocardiography, fluoroscopy, and haemodynamic monitoring that has increased the

safety of pericardiocentesis. Invariably echocardiography will have been used to diagnose or confirm the presence of an effusion, identify the best route of approach, and demonstrate successful drainage after the procedure. In an emergency, the patient's response to dehydrocholate may not be reliable. While dehydrocholate and circulation time measurement may be used as an adjunct to echocardiography during pericardiocentesis, it should not be used as an alternative.

Prevalence of hibernating myocardium in patients with severely impaired ischaemic left ventricles

EDITOR,—We read with interest the article by Al-Mohammad and colleagues on the prevalence of hibernating myocardium in patients with ischaemic left ventricular dysfunction.[1] Although we agree with the authors that positron emission tomography (PET) can provide very accurate information for the identification of hibernating myocardium, general statements regarding PET as being the "gold standard" for the diagnosis of viability might be misleading in view of recently published data.[2] In a particular subset of patients (those with severe postischaemic left ventricular dysfunction) PET is more accurate than other imaging techniques and, because of its technical characteristics, it is able to provide superior information on tissue viability. However, this requires the adoption of steady-state and standardised study conditions such as those achieved during hyperinsulinaemic euglycaemic clamp for an accurate quantification of the uptake of ^{18}F fluorodeoxyglucose (FDG) by the myocardium. In addition this method does not require the simultaneous measurement of myocardial blood flow for the assessment of viability.

Using this method we have shown that in patients with severe ischaemic heart failure, dobutamine stress echocardiography and PET have similar positive predictive values (68% and 66%) in the identification of hibernating myocardium, but that dobutamine stress echocardiography has a significantly lower negative predictive value than FDG-PET (54% *v* 96%; $p < 0.0001$).[3] This difference from previously published data reflects the study population in question who had severe left ventricular dysfunction. We have also shown that the baseline ejection fraction influences the predictive accuracy of PET, with the highest positive predictive accuracy in patients with an ejection fraction < 30%.[4]

We therefore advocate that in patients with postischaemic left ventricular dysfunction, myocardial viability is first sought by use of an easily available and inexpensive technique such as dobutamine stress echocardiography. If this is negative, then a more sensitive method of assessment should be used before ruling out coronary revascularisation. We feel that this should be quantitative PET using FDG during euglycaemic hyperinsulinaemic clamp.[5]

Regarding the assessment of myocardial blood flow to chronically dysfunctional but viable myocardium (hibernating) in patients with coronary artery disease, we believe that the use of ^{13}N ammonia as a flow tracer may lead to misleading pathophysiological hypotheses. The flow measurements obtained with this tracer reflect the average perfusion in a given mass of myocardium and are not weighted for the presence of scar. In contrast, myocardial blood flow assessed using ^{15}O labelled water mainly reflects flow to well perfused and hence viable tissue as the model used for the calculation assumes that the diffusion of water in scar tissue is negligible. Several papers have shown using PET with ^{15}O labelled water that the resting blood flow to hibernating myocardium is within normal limits. Moreover, similar conclusions have been reached using PET with ^{13}N ammonia in patients with hibernating myocardium, but without previous infarction.[6]

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1 Al-Mohammad A, Mahy IR, Norton MY, *et al.* Prevalence of hibernating myocardium in patients with severely impaired ischaemic left ventricles. *Heart* 1998;**80**:559–64.
2 Bax JJ, Wijns W, Cornel JH, *et al.* Accuracy of currently available techniques for prediction of functional recovery after revascularization in patients with left ventricular dysfunction due to chronic coronary artery disease: comparison of pooled data. *J Am Coll Cardiol* 1997;**30**:1451–60.
3 Pagano D, Bonser RS, Townend JN, *et al.* Predictive value of dobutamine echocardiography and positron emission tomography in identifying hibernating myocardium in patients with postischaemic heart failure. *Heart* 1998;**79**:281–8.
4 Ordoubadi F, Pagano D, Marinho NV, *et al.* Coronary revascularization in the treatment of moderate and severe postischemic left ventricular dysfunction. *Am J Cardiol* 1998;**82**:26–31.
5 Wijns W, Vatner SF, Camici PG. Hibernating myocardium. *N Engl J Med* 1998;**339**:173–81.
6 Camici PG, Wijns W, Borgers M, *et al.* Pathophysiological mechanisms of chronic reversible left ventricular dysfunction due to coronary artery disease (hibernating myocardium). *Circulation* 1997;**96**:3205–14.

This letter was shown to the authors, who reply as follows:

Barnes and Camici raise several interesting points. First, although dobutamine stress echocardiography and PET have comparable positive predictive accuracy in the assessment of myocardial viability, PET's superiority was maintained in terms of its negative predictive accuracy.[1] In addition, our study cohort had severe left ventricular impairment,[2] a group of patients for whom Barnes and Camici accept that PET is more accurate and provides superior information on tissue viability compared with other imaging techniques. Therefore, in the scientific search for the prevalence of hibernating myocardium in patients with severely impaired left ventricles, PET remains a superior research method. This, however, does not mean that PET should be used routinely in clinical practice. We share with Barnes and Camici the opinion that inexpensive and widely available techniques should be used first, while reserving the more sensitive PET for the negative cases.

Second, with regards to the use of euglycaemic hyperinsulinaemic clamp,[3] we accept that the ischaemic myocardium is insulin resistant.[4] Therefore, it might be expected that establishing a euglycaemic hyperinsulinaemic state (glucose clamp) may be associated with better detection of hibernating areas in which glucose uptake is normal or increased. In that respect, Knuuti *et al* found image quality was superior and fractional utilisation rates of ^{18}F FDG were twice as high during insulin clamp than after glucose loading ($p < 0.0001$).[5] However, this technique does not alter ^{18}F FDG uptake patterns in different myocardial areas compared to the standard glucose loading protocol.[5] Therefore, there is no evidence to suggest that obtaining superior image quality would have resulted in a different estimation of the prevalence of hibernating myocardium in the cohort of consecutive patients with severely impaired left ventricular contraction.

Third, while we acknowledge the results of perfusion studies using ^{15}O labelled water, Barnes and Camici accept that these could not be reproduced using ^{13}N ammonia in patients with previous infarction.[6] Most of our patients had documented myocardial infarction,[2] hence it was legitimate to use a widely tested and accepted definition of hibernating myocardium based on the experience gained from ^{13}N ammonia studies. In addition, recent studies confirm the reliability of ^{13}N ammonia as a perfusion marker and suggest its potential use as a predictor of viability.[7]

1 Pagano D, Bonser RS, Townend JN, *et al.* Predictive value of dobutamine echocardiography and positron emission tomography in identifying hibernating myocardium in patients with postischaemic heart failure. *Heart* 1998;**79**:281–8.
2 Al-Mohammad A, Mahy IR, Norton MY, *et al.* Prevalence of hibernating myocardium in patients with severely impaired ischaemic left ventricles. *Heart* 1998;**80**:559–64.
3 Wijns W, Vatner SF, Camici PG. Hibernating myocardium. *N Engl J Med* 1998;**339**:173–81.
4 Meeran K, Bloom SR. Lewis phenotypes, insulin resistance, and risk of ischaemic heart disease. *Br Heart J* 1994;**71**:305–6.
5 Knuuti MJ, Nuutila P, Ruotsalainen U, *et al.* Euglycemic hyperinsulinemic clamp and oral glucose load in stimulating myocardial glucose utilization during positron emission tomography. *J Nucl Med* 1992;**33**:1255–62.
6 Camici PG, Wijns W, Borgers M, *et al.* Pathophysiological mechanisms of chronic reversible left ventricular dysfunction due to coronary artery disease (hibernating myocardium). *Circulation* 1997;**96**:3205–14.
7 Kitsiou AN, Bacharach SL, Bartlett ML, *et al.* ^{13}N-ammonia myocardial blood flow and uptake: relation to functional outcome of asynergic regions after revascularization. *J Am Coll Cardiol* 1999;**33**:678–86.

Prognostic significance of electrical alternans *v* signal averaged ECG in predicting the outcome of electrophysiological testing and arrhythmia-free survival

EDITOR,—I was intrigued by the paper "Prognostic significance of electrical alternans versus signal averaged electrocardiography in predicting the outcome of electrophysiological testing and arrhythmia-free survival" by Armoundas *et al*.[1] In a time of statistics and prediction of important outcome events the reader has to pay much attention to the literature.

The conclusion "T wave alternans was a highly significant predictor of the outcome of electrophysiological testing and arrhythmia-free survival" is wrong; the positive predictive values are very low with very large confidence limits. The conclusion should be "There is a significant association between T wave alternans (TWA) and the outcome of electrophysiological (EP) testing. With single sided tests of significance there was a difference in arrhythmia-free survival for EP negative *v* EP positive patients, and for TWA negative *v* TWA positive patients."

There are also some possible literal errors and problematic calculations.

	Outcome	
Test	*+ve*	*−ve*
+ve	A	B
−ve	C	D

In the tables accuracy = (A + D)/n. In table 2 line 5 PV– is 80%; is this correct?

In table 3, are there three or four arrhythmic events? If there are three events it is impossible to recalculate the other parameters. Figures 2 and 3 show four events; in the text it states that three patients developed ventricular tachycardia or fibrillation.

In table 3 line 3 accuracy is 65% (calculated from (3 + 17)/31); n = 31 but the values compatible with your calculations are A = 3; B = 7; C = 1; D = 17; n = 28. The value of D is probably wrong: if n = 31 it should be 20; in this case other parameters will change.

With a little effort the authors could have illustrated the original numbers of outcome events, and not just the calculation of indices.

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1 Armoundas A, Rosenbaum DS, Ruskin JN, *et al*. Prognostic significance of electrical alternans versus signal averaged electrocardiography in predicting the outcome of electrophysiological testing and arrhythmia-free survival. *Heart* 1998;**80**:251–6.

This letter was shown to the authors, Dr Armoundis replies as follows:

The statement "T wave alternans was a highly significant predictor of the outcome of electrophysiological testing and arrhythmia-free survival" is an accurate characterisation of the results of our study as reflected by the p values for these two end points (0.0001 and 0.03).

The positive predictive values for T wave alternans as a predictor of the outcome of electrophysiological testing (64%) and 20 month arrhythmia-free survival (43%) are not low from a clinical perspective. For example, identification of a patient with a 43% probability of having cardiac arrest or sudden death within 20 months would certainly be adequate to justify therapeutic intervention. In contrast, the signal averaged ECG generally has been reported to have a positive predictive value on the order of 16%. It is true that our paper is an analysis of a small number of patients. We note that the T wave alternans results are confirmed in the larger group of patients in the original study[1] from which the patients in our study were drawn. The intriguing results of our pilot study merits prospective examination of the comparative predictive accuracy of T wave alternans compared with other risk stratifiers. Several recent prospective studies have reported confirmatory results.[2–4]

I am grateful to Dr Ector for detecting one transcription error and one arithmetic error. In table 2, the entry in the row labelled TWA + SAECG-II and the column labelled PV− should be changed from 80% to 89%. In table 3 in the row labelled SAECG-I, the entry in the column labelled PV+ should be changed from 30% to 23%, and in the column labelled RR it should be changed from 5.4 to 4.2. Correction of these errors, while small in magnitude, tends further to support the conclusions of our paper.

Table 3 is computed from predicted 20 month arrhythmia-free survival values as measured from the computed Kaplan-Meier curves. The number of predicted events were rounded to the nearest integer value in each stratum. This accounts for the variation in the total number of predicted events between 3 and 4, which may be imputed from the different rows of the table.

1 Rosenbaum DS, Jackson LE, Smith JM, *et al*. Electrical alternans and vulnerability to ventricular arrhythmias. *N Engl J Med* 1994;**330**:235–41.
2 Hohnloser SH, Klingenheben T, Yi-Gang L, *et al*. T wave alternans as a predictor of recurrent ventricular tachyarrhythmias in ICD recipients: prospective comparison with conventional risk markers. *J Cardiovasc Electrophysiol* 1998;**9**:1258–68.
3 Gold MR, Bloomfield DM, Anderson KP, *et al*. T wave alternans predicts arrhythmia vulnerability in patients undergoing electrophysiology study. *Circulation* 1998;**98**(suppl):I-647–8.
4 Klingenheben T, Cohen RJ, Peetermans J, *et al*. Predictive value of T wave alternans in patients with congestive heart failure. *Circulation* 1998; **98**(suppl):I-864.

Stenting for middle aortic syndrome

EDITOR,—Most of the patients in the article "Stenting for middle aortic syndrome" by Rajszys *et al* had long lesions with significant peak systolic gradients across the stenotic segment. Primary stenting of the lesions significantly reduced the peak systolic gradients.[1] We fail to understand the authors' preference for anticoagulants over ticlopidine. Studies have consistently shown that use of antithrombotic drugs are far superior to anticoagulants, not only in lowering the incidence of stent thrombosis but also in preventing bleeding complications.[2] The use of anticoagulants has largely been abandoned worldwide, even in coronary vessels, which have much smaller luminal diameters and lower flow rates compared with the aorta where the risk of thrombosis is low because of large lumen and higher flow rates.

Second, Rajszys *et al* deployed the Palmaz Schatz stents suboptimally to avoid overdilatation of aorta, repeating the procedure after the intimal tears have healed. Laplace's law theoretically places the aorta at an increased risk for rupture during angioplasty because less pressure is required to dilate the arterial walls as the diameter of the artery increases.[3] Hence, overdilatation of the aorta must never be attempted. Use of balloons sized 60–100% of the normal looking aorta and less than three times the maximally constricted segment have been shown to be safe and effective for aortoplasty.[4] Higher pressures are required in some cases for an optimal result. This is especially true for cases of middle aortic syndrome due to Takayasu arteritis (TA) where the vessel wall is thick and fibrosed. A similar strategy was used by Tyagi *et al* in their series of 38 cases of aortoplasty in TA including cases of middle aortic syndrome.[5] In our experience of de novo stenting of descending thoracic aorta in four cases of TA, we have shown that optimal deployment of Wall stents (Schneider Inc, Minneapolis, Minnesota, USA) using high pressure inflation (12–16 atm) could significantly increase the luminal diameters and abolish the peak systolic gradients.[6] We did not observe any case of stent thrombosis or any significant injury to the vessel wall. We feel that optimal deployment of stents would not only avoid stent thrombosis as occurred in one case in this series, but also avoid exposure to increased afterload and its adverse haemodynamic effects. It will also avoid the need for more procedures, limit fluoroscopic exposure, and prevent unnecessary hospital expenses. Even in children, stents can be safely deployed, taking into consideration the diameter of the normal aorta. These stents may be further dilated as the child grows.

Third, regarding the choice of stents: we feel that self expanding stents are preferable in long lesions of descending thoracic aorta to the Palmaz Schatz stent. They adapt to the anatomy of the aorta better and avoid deployment of several stents in long diffuse lesions.

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1 Rajszys GB, Qureshi SA, Ksiazyk J, *et al*. Middle aortic syndrome treated by stent implantation. *Heart* 1999;**81**:166–70.
2 Schomig A, Neumann FJ, Kastrati A, *et al*. A randomized comparison of antiplatelet and anticoagulant therapy after the placement of coronary artery stents. *N Engl J Med* 1996;**334**: 1084–9.
3 Yakes WF, Kumpe DA, Brown SB, *et al*. Percutaneous transluminal aortic angioplasty: techniques and results. *Radiology* 1989;**172**:965–70.
4 Lock JE, Bass JL, Amplatz K, *et al*. Balloon dilatation angioplasty of aortic coarctation in infants and children. *Circulation* 1983;**68**:109–16.
5 Tyagi S, Kaul UA, Nair M, *et al*. Balloon angioplasty of the aorta in Takayasu's arteritis: initial and long-term results. *Am Heart J* 1992;**124**: 876–82.
6 Bali HK, Jain S, Jain AK, *et al*. Stent supported angioplasty in Takayasu arteritis. *Int J Cardiol* 1998;**66**:S213–17.

NOTICES

Miami Children's Hospital will host the **third international symposium on paediatric cardiac intensive care,** 8–11 December 1999. The conference will be held at the Loews Hotel, Miami Beach, Florida, USA.

This three day symposium will emphasise aspects of perioperative care for children with congenital heart disease, and is designed for all physicians, nurses, and support personnel in cardiology, intensive care, cardiac surgery, cardiac anaesthesia, and neonatology who are involved in the care of critically ill neonates and children with heart disease.

For more details visit the conference website (www.mchpcs.com) or contact David Price & Associates, Inc at +1 305 663 6777; email: dpainc@compuserve.com.